on 2-ethylhexyl glucopyranoside. The results were that in the rat, 750 mg/kg/day represents the no-observed-toxic effect level (NOTEL) and 150 mg/kg/day represents the no-observed effect level (NOEL).

5. *Chronic toxicity.* Based on the NOTEL and NOEL results of the 28-day study conducted on 2-ethylhexyl glucopyranoside, there are no chronic health concerns.

6. *Animal metabolism.* Animal metabolism studies have not been conducted on 2-ethylhexyl glucopyranoside. However, structurally similar radiolabeled alkyl glucopyranosides were studied after oral administration to mice. The results indicate that the glycosidic bond was rapidly hydrolyzed in the intestine and liver to sugars and the parent alcohol. The sugars and alcohols then entered the pathways of lipid and carbohydrate metabolism.

7. *Metabolite toxicology.* The metabolites of 2-ethylhexyl glucopyranoside are expected to be the cleavage products at the glycosidic bond, 2-ethylhexanol and glucose. The toxicity of these two metabolites is well known.

8. *Endocrine disruption.* No evidence of endocrine disruption was observed in any of the studies conducted on 2-ethylhexyl glucopyranoside, nor are there any known reports of any estrogenic and adverse effects to human population as a result of the use of 2-ethyhexyl glucopyranoside.

C. Aggregate Exposure

1. *Dietary exposure.* Based on the metabolism study that indicates alkyl glucopyranosides are readily metabolized in the liver and intestine to glucose and the alcohol, exposure to 2-ethylhexyl glucopyranoside should not pose a dietary risk under any foreseeable circumstances to the U.S. population including infants and children.

i. *Food.* Exposures to 2-ethylhexyl glucopyranoside due to ingestion of food is not expected to occur.

ii. *Drinking water.* Exposures to 2-ethylhexyl glucopyranoside due to ingestion of water is not expected to occur.

2. *Non-dietary exposure.* Structurally similar alkyl glucopyranosides are currently being used in a number of institutional and household cleaning applications. These current uses are expected to result in significantly higher exposures than exposure due to the insignificant residue levels resulting from the use under the proposed exemption from the requirement of a tolerance applied to growing crops only.

D. Cumulative Effects.

From the results of the tests conducted on 2-ethylhexyl glucopyranoside, no evidence of any specific target organ toxicity has been produced. Therefore, there is no evidence of a common mechanism of toxicity with any other substance, and there is no reason to expect that the use of 2-ethyhexyl glucopyranoside will contribute to any cumulative toxicity resulting from exposures to other substances having a common mechanism of toxicity.

E. Safety Determination

1. *U.S. population.* The results of the acute, genotoxic, subacute and developmental toxicity studies conducted on 2-ethylhexyl glucopyranoside indicate a relatively low order of toxicity. Structurally similar alkyl glucopyranosides currently exempted from the requirement of a tolerance, also appear on EPA's List 4B Inert List. Therefore, due to the low order of toxicity of 2-ethylhexyl glucopyranoside and the lack of known adverse human health effects associated with this class of chemicals, the exemption from the requirement of a tolerance on growing crops only is not expected to result in any new, or adverse effects to human health or the environment.

2. *Infants and children.* Exposure to 2-ethylhexyl glucopyranosides to infants and children is not expected to occur. The substance will be used as an inert ingredient at low levels on growing crops only, and any residual levels are expected to be insignificant and consistent with structurally similar alkyl glucopyranosides currently exempted from the requirement of a tolerance.

F. International Tolerances

No codex maximum residue levels have been established for 2-ethyhexyl glucopyranoside.

[FR Doc. 02–19805 Filed 8–6–02; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2002–0151; FRL–7188–6]

Notice of Filing a Pesticide Petition to Establish a Tolerance for a Certain Pesticide Chemical in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of a pesticide petition proposing the establishment of regulations for residues of a certain pesticide chemical in or on various food commodities.

DATES: Comments, identified by docket ID number OPP–2002–0151, must be received on or before September 6, 2002.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I.C. of the **SUPPLEMENTARY INFORMATION**. To ensure proper receipt by EPA, it is imperative that you identify docket ID number OPP–2002–0151 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Sidney Jackson, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 305–7610; e-mail address: jackson.sidney@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be affected by this action if you are an agricultural producer, food manufacturer or pesticide manufacturer. Potentially affected categories and entities may include, but are not limited to:

Categories	NAICS codes	Examples of potentially affected entities
Industry	111 112 311 32532	Crop production Animal production Food manufacturing Pesticide manufacturing

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in the table could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether or not this action might apply to certain entities. If you have questions regarding the applicability of this action to a particular entity, consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

B. How Can I Get Additional Information, Including Copies of this Document and Other Related Documents?

1. *Electronically*. You may obtain electronic copies of this document, and certain other related documents that might be available electronically, from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select ''Laws and Regulations,'' ''Regulations and Proposed Rules,'' and then look up the entry for this document under the ''**Federal Register**—Environmental Documents.'' You can also go directly to the **Federal Register** listings at http://www.epa.gov/fedrgstr/.

2. *In person*. The Agency has established an official record for this actionunder docket ID number OPP–2002–0151. The official record consists of the documents specifically referenced in this action, any public comments received during an applicable comment period, and other information related to this action, including any information claimed as confidential business information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period, is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

C. How and to Whom Do I Submit Comments?

You may submit comments through the mail, in person, or electronically. To ensure proper receipt by EPA, it is imperative that you identify docket ID number OPP–2002–0151 in the subject line on the first page of your response.

1. *By mail*. Submit your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460.

2. *In person or by courier*. Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA. The PIRIB is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

3. *Electronically*. You may submit your comments electronically by e-mail to: opp-docket@epa.gov, or you can submit a computer disk as described above. Do not submit any information electronically that you consider to be CBI. Avoid the use of special characters and any form of encryption. Electronic submissions will be accepted in Wordperfect 6.1/8.0 or ASCII file format. All comments in electronic form must be identified by docket ID number OPP–2002–0151. Electronic comments may also be filed online at many Federal Depository Libraries.

D. How Should I Handle CBI That I Want to Submit to the Agency?

Do not submit any information electronically that you consider to be CBI. You may claim information that you submit to EPA in response to this document as CBI by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. In addition to one complete version of the comment that includes any information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public version of the official record. Information not marked confidential will be included in the public version of the official record without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person identified under **FOR FURTHER INFORMATION CONTACT**.

E. What Should I Consider as I Prepare My Comments for EPA?

You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

3. Provide copies of any technical information and/or data you used that support your views.

4. If you estimate potential burden or costs, explain how you arrived at the estimate that you provide.

5. Provide specific examples to illustrate your concerns.

6. Make sure to submit your comments by the deadline in this notice.

7. To ensure proper receipt by EPA, be sure to identify the docket ID number assigned to this action in the subject line on the first page of your response. You may also provide the name, date, and **Federal Register** citation.

II. What Action is the Agency Taking?

EPA has received a pesticide petition as follows proposing the establishment and/or amendment of regulations for residues of a certain pesticide chemical in or on various food commodities under section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that this petition contains data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

List of Subjects

Environmental protection, Agricultural commodities, Feed additives, Food additives, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: July 25, 2002.

Peter Caulkins,

Acting Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner summary of the pesticide petition is printed below as required by section 408(d)(3) of the Federal Food, Drug, and Cosmetic Act (FFDCA). The summary of the petition was prepared by the petitioner and represents the view of the petitioner. EPA is publishing the petition summary verbatim without editing it in any way. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

Interregional Reseaarch Project Number 4

PP 0E6205

Summary of Petitions

EPA has received a pesticide petition (PP 0E6205) from the Interregional Research Project Number 4 (IR-4), Technology Centre of New Jersey, Rutgers, the State University, 681 U. S. Highway #1 South, North Brunswick, NJ 08902 proposing, pursuant to section

408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR 180.300 by establishing a tolerance for residues of ethephon, (2-chloroethyl)phosphonic acid in or on the raw agricultural commodity coffee, bean at 0.5 parts per million (ppm). EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

This Notice was prepared by Aventis CropScience USA LP, Research Triangle Park, NC 27709.

A. Residue Chemistry

1. *Plant metabolism.* The qualitative nature of the residue in plants is adequately understood based on tomato, cantaloupe, apple, fig, pineapple, tobacco, grape, walnut, filbert, cherry, tangerine and lemon metabolism data. Ethephon degrades to ethylene, phosphate and chloride. Data indicate that proximal and distal translocation of ethephon to fruits may occur following application to leaves. The residue of concern in plants is ethephon.

2. *Analytical method.* Adequate methods for purposes of enforcement of ethephon tolerances in plant commodities, ruminant tissues, and milk are available. The Amchem-Plant Method (PAM, Vol. II, Method I) is the recommended method for enforcement purposes for plant commodities and processed products other than wheat and barley straw. The Amchem-Cereal Method (forwarded to the Food and Drug Administration (FDA) for inclusion in the PAM, Vol. II, Method I) is the recommended method for enforcement purposes for wheat and barley straw. The Union Carbide-Animal Method (forwarded to the FDA for inclusion in the PAM, Vol. II, Method III) is the recommended method for enforcement purposes for milk and animal tissues. These methods employ diazomethane as a methylating agent. A new plant and animal method has been submitted for enforcement purposes that does not employ diazomethane. The method principally involves the decomposition of ethephon to ethylene to determine the residues of ethephon. An independent lab validation of this method has been completed and accepted by EPA.

3. *Magnitude of residues.* Residue studies have been conducted to support ethephon registrations on: cotton, apple, cherry, tomato, wheat, barley, pepper, grape, tobacco, walnut, almond, blackberry, cantaloupe, pineapple, sugarcane and macadamia nuts. In addition, IR-4 has conducted residue studies to support use on coffee. All residue data requirements cited in the ethephon Reregistration Eligibility Document (RED) have been submitted to EPA. As a result of this work, increased tolerances have been proposed for cottonseed (6 ppm, PP 6F4743) and cotton gin by-products (180 ppm, amendment to PP 1H5603). As part of the reregistration process, the following tolerances will be revoked: cucumber, filbert, lemon, pineapple forage and fodder, pumpkin, tangerine, tangerine hybrids and sugarcane molasses. The tolerances for residues of ethephon in or on food and feed commodities are currently based in terms of ethephon per se. Processing studies have been conducted on apple, barley, cottonseed, grape, pineapple, tomato, and wheat and are deemed adequate to determine the extent to which residues of ethephon concentrate in food/feed items upon processing of the raw agricultural commodity. Data indicate that ethephon residues concentrate in apple juice, dried apple pomace, barley hulls, cottonseed meal, grape juice, raisin, raisin waste, dried grape pomace, pineapple bran and pulp, dried tomato pomace, wheat bran, wheat shorts and germ and red dog. Available apple processing data indicate that residues of ethephon do not concentrate in wet apple pomace. Therefore, a feed additive tolerance on apple pomace is not required. Available tomato processing data indicate that residues of ethephon do not concentrate in tomato paste and, therefore, no tolerance is needed. Pineapple processing data indicate that residues of ethephon concentrate in dried pineapple bran (5.3X; no longer a processed commodity) and wet pulp (1.2X), but do not concentrate in juice, syrup, and slices. No feed additive tolerance for residues of ethephon in processed pineapple is required. As a result of a recent cow feeding study, new animal tolerances have been proposed. The following tolerances have been proposed for cattle, goat, hog, horse, and sheep: meat - 0.02 ppm; meat byproducts (except kidney) - 0.20 ppm; kidney - 1.0 ppm; fat 0.02 ppm, and milk (cow and goat) - 0.01 ppm. Following a hen feeding study, new tolerances were proposed for poultry: poultry meat - 0.01 ppm; poultry meat byproducts (except liver) - 0.01 ppm; poultry fat - 0.02 ppm; poultry liver - 0.05 ppm; and eggs - 0.002 ppm.

B. Toxicological Profile

1. *Acute toxicity.* A complete battery of acute toxicity studies for ethephon technical was completed. The acute oral toxicity study resulted in a lethal dose LD_{50} of 1,600 milligram/kilogram (mg/kg) for both sexes. The acute dermal toxicity in rabbits resulted in an LD_{50} in either sex of greater than 5,000 mg/kg. The acute inhalation study in rats resulted in a lethal concentration LC_{50} of 4.52 milligram/liter (mg/l). Ethephon was corrosive to the skin of rabbits in the primary dermal irritation study. Therefore, the primary eye irritation study in rabbits was not required. The dermal sensitization study in guinea pigs indicated that ethephon is not a sensitizer. Based on the results of the dermal irritation study, and the anticipated results in an eye irritation study, ethephon technical is placed in toxicity Category I. Based on the acute toxicity data cited above, the registrant concluded that ethephon technical does not pose any acute dietary risks.

2. *Genotoxicty.* The potential for genetic toxicity of ethephon was evaluated in several assays. The compound was found to be mutagenic in strain TA-1535 with and without S9 activation in the Ames assay. In the *in vitro* chromosomal aberrations study with Chinese hamster ovary cells, ethephon was negative. Ethephon was tested for unscheduled DNA synthesis in the rat hepatocyte system and was found to be negative. Based on the data cited above, Aventis contends that the weight of evidence indicates that ethephon technical does not pose a risk of mutagenicity or genotoxicity.

3. *Reproductive and developmental toxicity.* Ethephon has been tested for reproductive toxicity in rats and developmental toxicity in both rats and rabbits (two studies in each species). The results of these studies are summarized below:

i. In a two generation reproduction study, 28 Sprague-Dawley rats per sex per dose were administered 0, 300, 3,000, or 30,000 ppm (0,15, 150, or 1,500 mg/kg/day of ethephon in the diet. For the offspring, a no observed adverse effect level (NOAEL) of 15 mg/kg/day and a lowest observed adverse effect level (LOAEL) of 150 mg/kg/day was established based on decreased body weight gain in the females at 150 mg/kg/day and in both sexes at 1,500 mg/kg/day. No effects were observed on fertility, gestation, mating, organ weights, or histopathology in any generation.

ii. In rats, ethephon was administered by gavage at doses of 0, 20, 600, or 1,800 mg/kg for gestation days 6 through 15.

At 1,800 mg/kg/ day, 14 of the 24 treated female rats died. No toxic effects were observed at lower doses. The NOAEL for maternal and developmental toxicity was 600 mg/kg/day. In a second study, rats were dosed by gavage at 0, 125, 250, or 500 mg/kg/day on days 6 through 15 of gestation. No toxic effects were observed at any dose. The NOAEL for maternal and developmental toxicity was 500 mg/kg/day.

iii. In rabbits, ethephon was administered by gavage at doses of 0, 50, 100, and 250 mg/kg for gestation days 6 through 19. The number of doses with live fetuses were 10, 12, 8, and 5, respectively. Resorptions were increased at 100 mg/kg/day and statistically significantly increased at 250 mg/kg/day. At 250 mg/kg/day, does were depressed, ataxic, showed an increase of clinical observations and gross pathology in the gut. The NOAEL for maternal toxicity was 50 mg/kg/day and the NOAEL for developmental toxicity was 50 mg/kg/day. In a second study, rabbits were dosed by gavage at 0, 62.5, 125, or 250 mg/kg/day on days 6 through 19 of gestation. Maternal morbidity, mortality, and clinical signs of toxicity were observed at 250 mg/kg/day. Fetal toxicity, consisting of decreased number of live fetuses per doe, increased early resorptions and post implantation loss was observed at 250 mg/kg/day. A NOAEL for maternal and developmental toxicity of 125 mg/kg/day was observed.

Based on the 2-generation reproduction study in rats, ethephon is not considered a reproductive toxicant and shows no evidence of endocrine effects. The data from the developmental toxicity studies on ethephon show no evidence of a potential for developmental effects (malformations or variations) at doses that are not maternally toxic. The NOAEL for both maternal and developmental toxicity in rats was 500 mg/kg/day and for rabbits, the NOAEL for both maternal and developmental toxicity was 50 mg/kg/day, respectively.

4. *Subchronic toxicity.* The subchronic toxicity of ethephon has been studied in three human studies and a 21–day dermal study in rabbits. These studies are summarized below:

i. Male and female subjects received ethephon at doses of 0.17 and 0.33 mg/kg/day for 22 days. The daily doses were divided into 3 gelatin capsules. No adverse effects were noted in clinical observations, hematology, serum chemistry including red blood cell cholinesterase inhibitors (RBC ChE) and urinalysis. There was a significant decrease in plasma ChE for both treatment groups, although the effect at 0.17 mg/kg/day appeared to be very close to the threshold for significance.

ii. Male and female subjects received ethephon at a dosage of 0.5 mg/kg/day for 16 days. The daily dose was divided into 3 gelatin capsules. No adverse effects were noted in clinical observations, hematology, serum chemistry (including RBC ChE) and urinalysis. There was a significant decrease in plasma cholinesterase.

iii. Ethephon was administered to male and female subjects at a daily dose of 124 mg/day (1.8 mg/kg/day average for both sexes) divided up into 3 gelatin capsules for 28 days. Clinical signs of toxicity were observed and included diarrhea, urgency of bowel movements, urinary urgency and stomach cramps. No effects were noted with regard to hematology, urinalysis or serum chemistry including cholinesterase evaluations.

iv. In a 21–day dermal study, 10 rabbits per sex per group were dosed dermally at 0, 25, 75, and 150 mg/kg/day, 5–days per week for 3 weeks. Skin effects were observed at all doses. Effects ranged from erythema and desquamation at the lowest dose to acanthosis and chronic inflammation at 150 mg/kg/day. No systemic treatment-related effects were observed on body weight, food consumption, organ weight or histopathology. The systemic NOAEL was greater than 150 mg/kg/day.

Based on the results of the three studies in humans, a LOAEL of 1.8 mg/kg/day was established in the 28–day study. In the 22–day study, 0.17 mg/kg/day appeared to be very close to the threshold for significance. The systemic NOAEL in the 21–day dermal study in rabbits was greater than 150 mg/kg/day.

5. *Chronic toxicity.* A 2 year chronic toxicity/carcinogenicity study in rats, an 18-month mouse carcinogenicity study, a 1–year study in dogs, and a 2–year chronic study in dogs were performed on ethephon technical. These studies are summarized below:

i. A combined chronic/carcinogenicity study was performed on ethephon in Sprague-Dawley rats. Doses administered in the feed were 0, 300, 3,000, 10,000, or 30,000 ppm for 95 weeks to the males and 103 weeks for the females. The doses administered relative to body weight were 0, 13, 131, 446, or 1,416 mg/kg/day for males and 0, 16, 161, 543, or 1,794 mg/kg/day for females. Plasma and erythrocyte cholinesterase was inhibited at all doses (NOAEL <300 ppm). Brain cholinesterase inhibition was not observed. A decrease in male body weight was observed at 10,000 ppm. At 30,000 ppm a body weight decrease was observed in both sexes. Additional effects at 30,000 ppm were thyroglossal duct cysts, kidney glomerulo-sclerosis, nephritis, and biliary hyperplasia cholangiofibrosis. No carcinogenic effects were observed.

ii. Male and female CD-1 mice were administered ethephon in the diet at 0, 100, 1,000, or 10,000 ppm (0, 15.5, 156, or 1,630 mg/kg/day) for 78 weeks. An additional dose level of 50,000 ppm was terminated at 12-weeks because of excessive morbidity and mortality. No evidence of treatment related tumors was observed. A NOAEL of 15.5 mg/kg/day was determined for plasma cholinesterase inhibition. At 1,630 mg/kg/day male body weights were increased and female body weights decreased compared to controls.

iii. Ethephon technical was administered in the feed at 0, 30, 300, and 3,000 ppm (0, 0.75, 7.5, or 75 mg/kg/day) to male and female beagle dogs for 2 years. Due to toxicity/morbidity, the high dose was reduced as follows: 75 mg/kg/day weeks 0–3; 50 mg/kg/day weeks 4–5; 25 mg/kg/day weeks 6-24; 37.5 mg/kg/day weeks 25-104. Plasma cholinesterase was inhibited at all doses (NOAEL< 0.75 mg/kg/day). A NOAEL for erythrocyte cholinesterase inhibition of 0.75 mg/kg/day with a LOAEL of 7.5 mg/kg/day was observed. Histopathology showed smooth muscle atrophy in the gut at 7.5 mg/kg/day with a NOAEL of 0.75 mg/kg/day.

iv. Ethephon was administered in the feed at doses of 0, 100, 300, 1,000, or 2,000 ppm (0, 2.7, 8.2, 28.5, or 52.1 mg/kg/day) to male and female beagle dogs for 52 weeks. A systemic NOAEL of 1,000 ppm (28.5 mg/kg/day) was observed for decreased spleen weight, body weight, hemoglobin and hematocrit in males. The females showed a decreased spleen/body weight ratio for the same NOAEL. Cholinesterase inhibition was not determined.

The NOAEL in the chronic rat study was 131 mg/kg/day based on the decreased body weight gains in males. The NOAEL in the most recent 1–year dog study was determined to be 28.5 mg/kg/day based on body weight, organ weight effects and hematology effects. Ethephon has been tested in both rats and mice for carcinogenic activity. No carcinogenic effects were observed.

6. *Animal metabolism.* The rat metabolism study consisted of a single intravenous dose group at 50 mg/kg, and single and multiple oral high dose groups at 50 and 1,000 mg/kg. The oral Cmax (maximum concentrations) were reached at 1.3 and 1 hours for the 50 mg/kg dose and 1.9 and 2.5 hours for the 1,000 mg/kg dose in males and females, respectively. The $t_{1/2}$ of the

rapid excretion phase (A-phase) at the 50 mg/kg dose was 7 hours for both sexes and 4 and 9 hours at 1,000 mg/kg for the males and females, respectively. Oral and intravenous doses were rapidly excreted in the urine and accounted for 48 to 71% of the administered radioactivity. Approximately 7% was excreted in the feces. Exhaled ethylene was 10–20% and CO2 was less than 1% of the administered dose. The highest tissue concentrations were found in the blood, bone, liver, kidney, and spleen with no significant differences between single and multiple dosing. No significant differences were observed in the excretion pattern with either sex or multiple dosing.

In a goat metabolism study, ethephon was incorporated into natural products (glutathione conjugates, protein, glycogen, and triglycerides) and expired as CO2 and ethylene.

In a hen metabolism study, ethephon metabolism involved an initial removal of chlorine to form 2-hydroxyethanephosphonic acid followed by further metabolism which results in the release of ethylene and carbon dioxide as well as intermediates which can enter into fundamental biochemical pathways leading to the biosynthesis of proteins and lipids. Aventis believes that ethephon technical is not metabolized to breakdown products that can be reasonably expected to present any chronic dietary risk.

7. *Metabolite toxicology.* Ethephon degrades to ethylene phosphate and chloride. Therefore, no significant toxicity is anticipated from these breakdown/metabolites.

8. *Endocrine disruption.* EPA is required under the FFDCA, as amended by Federal Quality Protection Act (FQPA), to develop a screening program to determine certain substances (including all pesticide active and other ingredients) ''may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen, or other such endocrine effects as the Administrator may designate.'' Following the recommendations of its Endocrine Disruptor Screening and Testing Advisory Committee (EDSTAC), EPA determined that there were scientific bases for including, as part of the program, the androgen and thyroid hormone systems, in addition to the estrogen hormone system. EPA also adopted EDSTAC's recommendation that the Program include evaluations of potential effects in wildlife. For pesticide chemicals, EPA will use FIFRA and, to the extent that effects in wildlife may help determine whether a substance may have an effect in humans, FFDCA authority to require the wildlife evaluations. As the science develops and resources allow, screening of additional hormone systems may be added to the Endocrine Disruptor Screening Program (EDSP). When the appropriate screening and/or testing protocols being considered under the Agency's EDSP have been developed, ethephon may be subjected to additional screening and/or testing to better characterize effects related to endocrine disruption.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food.* Ethephon is registered for use on the following food crops: cotton, apple, cherry, tomato, wheat, barley, pepper, grape, tobacco, walnut, almond, blackberry, cantaloupe, pineapple, sugarcane, and macadamia nuts. In addition, IR-4 has conducted work to support new use on coffee. Ethephon has several ornamental/non-food applications as well. All residue requirements cited in the ethephon RED have been submitted to EPA. As a result of this work, increased tolerances have been proposed for cottonseed (6 ppm, PP 6F4743) and cotton gin byproducts (180 ppm, amendment to PP 1H5603). As part of the reregistration process, the following tolerances will be revoked: cucumber, filbert, lemon, pineapple, forage, fodder, pumpkin, tangerine, tangerine hybrids, and sugarcane molasses. The tolerances for residues of ethephon in or on food and feed commodities are currently based in terms of ethephon per se. An enforcement method was submitted to EPA for determination of residues of ethephon in/on plant commodities and in milk, ruminant and poultry tissues. The ethephon RED lists the number of treated acres by crop for all major ethephon uses in the United States.

ii. *Drinking water.* Based on the available studies and the use pattern, Aventis does not anticipate residues of ethephon in drinking water. There is no established Maximum Concentration Level or Health Advisory Level for ethephon under the Safe Drinking Water Act.

2. *Non-dietary exposure.* The potential for non-occupational exposure to the general public is also insignificant since only approximately 800 lbs of ethephon technical is sold in the U.S. home and garden market annually. The residential lawn or garden uses anticipated for these products where the general population may be exposed via inhalation or dermal routes are negligible. The home and garden formulation that is sold in the United States contains only 3.9% ethephon which would further limit exposure.

D. Cumulative Effects

While ethephon is an inhibitor of ChE of the plasma and RBC, it has not demonstrated any ability to inhibit brain ChE in rats, mice, or dogs under condition of a chronic dietary dosing regimen. Furthermore, unlike classic organophosphate ChE inhibitors, ethephon did not induce symptoms of ChE inhibition, such as constriction of the pupils, salivation, lacrimation, diarrhea, urination, tremors, and convulsions under chronic feeding of doses up to 30,000, 10,000, and 2,000 ppm in the rat, mouse, and dog, respectively. In the rat study, the plasma and RBC ChE were inhibited approximately 55% and 85%, respectively. In the mouse study, both peripheral ChEs were inhibited by approximately 70%. Although cholinesterase determinations were not performed in the 1 year dog study, in a 2 year dog study, plasma and RBC ChE were inhibited 60% and 70%, respectively. Despite these high degrees of inhibition of peripheral ChE, no clinical signs or symptoms consistent with ChE inhibition occurred in these studies. It is generally only under very extreme conditions such as high doses administered via oral gavage or under occlusive dermal dressing in rabbits in which signs that are consistent with ChE inhibition are observed. These clinical signs generally occur at doses that produce acute lethality. However, these signs may in fact be unrelated to CNS ChE inhibition and could be a non-specific reaction to the acidic and, therefore, highly irritant nature of ethephon.

Ethephon should not be regarded as a classical inhibitor of ChE such as the carbamates and organophosphates since it does not produce the typical nervous system effects of those compounds. The recently updated chronic data base adequately proves that very high dietary doses of ethephon do not inhibit brain ChE, that it does not produce the classical clinical signs of ChE inhibition, and that it does not produce life-shortening effects, despite moderate to severe lifetime inhibition of both plasma and RBC ChE. The inhibition of ChE by ethephon is only an indicator of exposure and is not a measure of its potential for inducing ChE-mediated toxicity. In summary, Aventis concludes that consideration of a common mechanism of toxicity is not appropriate at this time since there is no significant toxicity observed for ethephon. Even at high doses, ethephon does not act as a classical inhibitor of cholinesterase.

Exposure, even at high doses, does not lead to brain cholinesterase inhibition. There is no reliable data to indicate that the effects noted would be cumulative with those of organophosphate or carbamate-type compounds. Therefore, Aventis has considered only the potential risks of ethephon in its exposure assessment.

E. Safety Determination

EPA reference dose (RfD) Peer Review Committee determined that the RfD should be based on the 28–day study in humans. Using the LOAEL of 1.8 mg/kg/day in this study and an uncertainty factor (UF) of 100 to account for intraspecies variability and the lack of a NOAEL, an RfD of 0.018 mg/kg/day was established as the chronic dietary endpoint.

1. *U.S. population.* A chronic dietary risk assessment which included all proposed changes in ethephon tolerances was conducted on ethephon using two approaches: A Tier 1 approach using tolerance-level residues for all foods included in the analysis, and Monte Carlo simulations using tolerance-level residues for all foods adjusted for percent crop treated (PCT) (Tier 3). Using the Tier 1 approach, margin of exposure (MOEs) at the percentiles of exposure for the overall U.S. population were 25 and 9, respectively. Using Tier 3 procedures in which residues were adjusted for the PCT, MOEs were 114 and 42, respectively. Acute exposure was also estimated for infants and children 1 to 6 years of age. In the Tier 1 analysis, the most highly exposed subgroup was infants. For this population, MOEs at the 95th and 99th percentiles of exposure were 7 and 4, respectively. Using the Tier 3 method MOEs were 56 and 12, respectively. Even under the conservative assumptions presented here, the more realistic estimates of dietary exposure (Tier 3 analyses) clearly demonstrate adequate MOEs up to the 99th percentile of exposure for all population groups analyzed.

2. *Infants and children.* In assessing the potential for additional sensitivity of infants and children to residues of ethephon, the available developmental toxicity and reproductive toxicity studies and the potential for endocrine modulation by ethephon were considered. Developmental toxicity studies in two species indicate that ethephon is not a teratogen. The 2 generation reproduction study in rats demonstrated that there were no adverse effects on reproductive performance, fertility, fecundity, pup survival, or pup development. Maternal and developmental NOAELs and LOAELs were comparable, indicating no increase in susceptibility of developing organisms. No evidence of endocrine effects were noted in any study. It is therefore, concluded that ethephon poses no additional risk for infants and children and no additional uncertainty factor is warranted. FFDCA section 408 provides that an additional safety factor for infants and children may be applied in the case of threshold effects. Since, as discussed in the previous section, the toxicology studies do not indicate that young animals are any more susceptible than adult animals and the fact that the proposed RfD calculated from the LOAEL from the 28-day human study already incorporates an additional uncertainty factor, Aventis believes that an adequate margin of safety is, therefore, provided by the RfD established by EPA. Additionally, this LOAEL is also 8X lower than the next lowest NOAEL (2 generation reproduction study, NOAEL=15 mg/kg/day) in the ethephon toxicology data base. Ethephon has no endocrine-modulation characteristics as demonstrated by the lack of endocrine effects in developmental, reproductive, subchronic, and chronic studies.

An RfD of 0.018 mg/kg/day has been established by EPA based on the LOAEL in the 28–day human study. Adequate MOEs exist for all populations including infants and children. No additional uncertainty factor for infants and children is warranted based on the completeness and reliability of the database, the demonstrated lack of increased risk to developing organisms, and the lack of endocrine-modulating effects.

F. International Tolerances

The codex maximum residue limits (MRLs) for grape is 10 mg/kg verses 2 ppm for U.S. tolerance. The tomato codex MRL is 3 mg/kg verses 2 ppm for the U.S. tolerance. All other U.S. tolerances are identical to corresponding codex MRLs.

[FR Doc. 02–19803 Filed 8–6–02; 8:45 am]

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ENVIRONMENTAL PROTECTION AGENCY

[OPP–2002–0173; FRL–7191–3]

Notice of Filing a Pesticide Petition to Establish a Tolerance for a Certain Pesticide Chemical in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of a pesticide petition proposing the establishment of regulations for residues of a certain pesticide chemical in or on various food commodities.

DATES: Comments, identified by docket ID number OPP–2002–0173, must be received on or before September 6, 2002.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I.C. of the **SUPPLEMENTARY INFORMATION**. To ensure proper receipt by EPA, it is imperative that you identify docket ID number OPP–2002–0173 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Sidney Jackson, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460;telephone number: (703) 305–7610; e-mail address: jackson.sidney@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be affected by this action if you are an agricultural producer, food manufacturer or pesticide manufacturer. Potentially affected categories and entities may include, but are not limited to:

Categories	NAICS codes	Examples of potentially affected entities
Industry	111 112 311 32532	Crop production Animal production Food manufacturing Pesticide manufacturing

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in the table could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether or not this action might apply to certain entities. If you have questions regarding the applicability of this action to a particular entity, consult the person listed under **FOR FURTHER INFORMATION CONTACT**.